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## DISCUSSION

**Dr Julie Ann Freischlag** (*Baltimore, Md*). Could you define how a myocardial infarction was determined, and what you required in order to make that diagnosis?

**Dr Jessica P. Simons.** The way that the data capture postoperative myocardial ischemia are categorized as none, troponin elevation only, or clinical or EKG evidence of MI.

Myocardial infarction is defined at the discretion of the surgeon, ultimately, when they fill out the data sheets, but it includes either clinical evidence of myocardial infarction, such as chest pain or EKG changes consistent with an MI.

**Dr Jon Matsumura** (*Madison, Wisc*). What are the criteria to screen a patient with a troponin? Were you only testing patients who had symptoms, or was it all the patients in the database had a routine troponin? With a prophylactic operation for aneurysm repair or asymptomatic carotid, predicting the long-term survival is helpful for the patient decision making.

**Dr Simons.** I think that's a really excellent point, and that it would be really helpful to know what the indication was. We don't have that data available to us, why a patient would have had troponins checked postoperatively. It's not a routine part of the management of all patients in the VSGNE, but rather, it's at the discretion of the surgeon.

**Dr Michael Conte** (*San Francisco, Calif*). I think that your slide where you contrasted the implications about causality versus association is really the most important point, because I think, again, this information may be misinterpreted in light of the types of conversations that surround it; for example, the CREST end point.

And, unfortunately, your conclusion slide actually erred back in the wrong direction by suggesting causality, the way I read it, when you said that these postoperative events were themselves directly related to later mortality. I think if you took 1000 cardiovascular patients in the clinic and gave them all a stress test, it would be no shock to find out that the ones that failed the stress test would not have quite as good long-term survival as those that passed the stress test. But, the stress test doesn't cause the death. That is entirely analogous to the issue with the majority of postoperative MIs.

I think that this becomes a critical issue with interpretation of the findings, while I concur with the importance of working to prevent these events. My major question to you is: did you look at whether or not all of the patients were treated according to guidelines with cardioprotective medications? Because I think that really is the bottom line for perioperative management. It's not the procedure that's causing the late mortality, it's really whether or not they are getting appropriate medical care before, during, and after.

**Dr Simons.** We did take a look to some extent at that, although I think that that question is being addressed a little bit

more thoroughly with another VSGNE study. But, in brief, we did find that the use of statin and antiplatelet agents at the time of discharge was protective against death in our Cox proportional hazard model, suggesting exactly as you're saying, that if patients are treated medically more appropriately, then perhaps that would be what impacts survival over the long term.

**Dr John Chang** (*Roslyn, NY*). What would be your suggestions in terms of optimization preoperatively? Do you think that everybody who has a positive stress test should go through cardiac catheterization or revascularization?

**Dr Simons.** Well, I think we probably need to lean on our cardiology colleagues a little bit more in order to figure out exactly what the right things would be to better preoptimize patients preoperatively and medically manage them postoperatively as well. Certainly the data suggest a benefit to medications such as statins and antiplatelet agents for improving survival. But, beyond that, who needs a stress test and who needs preoperative cardiac catheterization is a question I would work with our cardiology colleagues to better answer.

**Dr Matthew Eagleton** (*Cleveland, Ohio*). When you're looking at cause of death at 5 years, do you have any categorization as to cause of death, such as cardiovascular death versus cancer death?

**Dr Simons.** We don't have that data available to us in this data set.

**Dr Cheong Lee** (*Milwaukee, Wisc*). Elevation in troponin can be also related to impairment in renal function, so do you think this is actually a reflection of renal impairment that you're seeing the elevations in troponin being related with mortality?

**Dr Simons.** I think it's a little hard to know for sure. I know that looking through our data, the percentage of patients who have significant renal disease—which is defined by a creatinine greater than 1.8—is very small, so I suspect that that influence would be very minor. But, that is a reasonable point.

The other confounding issue is whether troponins were checked disproportionately among those patients with renal failure, which would bias the data. So, I can't really answer that question specifically.

**Dr Amy Reed** (*Hershey, Pa*). I'm assuming this is from the VSGNE database. And, I know on a VQI, we do put down whether there has been any preoperative evaluation. Was that not available in this data set that you aren't aware of that?

**Dr Simons.** No, we did have access to that variable. And, we did include in some of our analyses whether or not a preoperative stress test was performed. And, it was not performed for the majority of patients. I think it was positive only in an extremely small percentage, as you would expect to find. Regardless, it ultimately didn't end up being significant on univariate screening, and that's why it didn't make it into the final analysis.